

# Oesophageal Electrocardiography in the Study of Cardiac Arrhythmias

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Precise knowledge of atrial activity and its temporal relation to the QRS complex is the key to the diagnosis of many cardiac arrhythmias. In this respect the value of the oesophageal electrocardiographic lead is well established and depends on the recording of large distinctive P waves which may be barely discernible or absent in other leads (Bussan, Torin, and Scherf, 1955; Schrire and Vogelpoel, 1955; Kistin and Bruce, 1957). Previous studies of cardiac arrhythmias by the use of oesophageal leads either aimed to emphasize the limitations of the standard leads (Brown, 1936; Kistin and Landowne, 1951; Prinzmetal *et al.*, 1952; Bellet, 1963), or were primarily concerned with the unravelling of complex arrhythmias with the report of a few illustrative electrocardiograms (Kistin and Bruce, 1957; Copeland, Tullis, and Brody, 1959; Rodensky and Wasserman, 1962). The present study represents a critical evaluation of the capabilities of the oesophageal lead in the whole field of cardiac arrhythmias.

## SUBJECTS AND METHODS

Studies were made on 148 subjects with various disturbances of the cardiac rhythm, and 242 oesophageal electrocardiograms were recorded. Some of the cases represent arrhythmias induced during cardiac catheterization. The various abnormalities encountered are listed in the Table. The oesophageal electrocardiogram was recorded through a Wilson unipolar connexion (VE lead). The optimum position of the oesophageal electrode was determined by scanning tracings obtained from several levels. The position yielding the best P wave for study varies with the individual and with the contour and direction of the QRS and T waves. The VE lead was always recorded simultaneously with one or more of the standard leads, usually lead II. The illustrated cardiograms were recorded at a paper speed

of 25 mm./sec. on a 2- or 4-channel direct-writing electrocardiograph (Cardiopan-2 and Minograph-42 B). The number under VE in the illustrations designates the position of the oesophageal electrode in centimetres from the nares.

TABLE  
CLASSIFICATION OF ABNORMALITIES  
ENCOUNTERED IN 242 OESOPHAGEAL LEAD  
SURVEY

Abnormalities	No. of cases
Sinus tachycardia	5
Sinus bradycardia	6
Sinus arrhythmia	10
Sino-atrial block	4
Atrioventricular dissociation	8
Atrioventricular heart block	
Incomplete (dropped beats)	10
Complete	5
Premature systoles	
Atrial } supraventricular	49
Nodal }	
Bundle of His	1
Ventricular	65
Paroxysmal tachycardias	
Sinus with AV heart block	1
Atrial } supraventricular	10
Nodal }	
Atrial with AV heart block	8
Ventricular	35
Atrial flutter	5
Atrial fibrillation	20

## RESULTS

The VE lead has proved valuable in unravelling the exact nature of the following cardiac arrhythmias: supraventricular premature systoles, paroxysmal atrial tachycardia with block, tachycardias of supraventricular origin with aberration of the ventricular complex, AV dissociation, ventricular premature systoles, ventricular tachycardia, and rare cardiac arrhythmias.

*Supraventricular Premature Systoles.* The upper, middle, and lower strips (Fig. 1 a, b, and c) were

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recorded from 3 different patients. Lead II in the upper strip shows two aberrant ventricular complexes (the third and the fifth). These could be interpreted as ventricular premature systoles; however, inspection of the T waves of the preceding beats reveals a slight deformity which is more evident before the first aberrant ventricular complex, and is caused by the superimposition of a premature P wave. The VE lead, on the other hand, clearly demonstrates the presence of two premature P waves preceding the aberrant ventricular complexes. It also illustrates that the two premature P waves arise from two different foci.

In the middle strip (b), the fourth, fifth, and sixth ventricular complexes in lead II show aberration which is marked in the fourth and slight in the fifth and sixth beats. There are no discernible P waves preceding these three ventricular complexes. The VE lead, on the other hand, demonstrates the presence of a series of three premature P waves.

In the lower strip (c), the fourth ventricular complex is a supraventricular premature systole. The premature P wave is fused with the T wave of the preceding beat, which is rendered slightly higher and peaked. The sixth ventricular complex is aberrant, and could have been interpreted as an interpolated ventricular premature systole, but the VE lead demonstrates the presence of two premature P waves with marked aberration of the first ventricular complex and minimal aberration of the second.

*Comment.* Aberrant conduction of the supraventricular premature systole is a common phenomenon (Scherf and Schott, 1953). It was claimed that more of these premature systoles were followed by abnormal ventricular complexes than by normal ones (Berliner and Lewithin, 1945). Aberration is most apt to occur when the premature systoles fall in early diastole, because they then find the conduction system and ventricular myocardium still partially refractory (Bellet, 1963). However, other factors are important. In this connexion, the observation seen in (b) and (c), namely that the first of a series of supraventricular premature systoles shows a particularly delayed conduction and marked degree of aberration, has been reported by others (Scherf and Schott, 1953). The beats occurring after long intervals have a long refractory period so that a premature systole following on such a beat will find the tissue only partially recovered; this will account for both delayed AV as well as for aberrant intraventricular conduction (Scherf and Schott, 1953). If the degree of aberration of the premature systoles is marked and if the premature P wave is buried in the T wave of

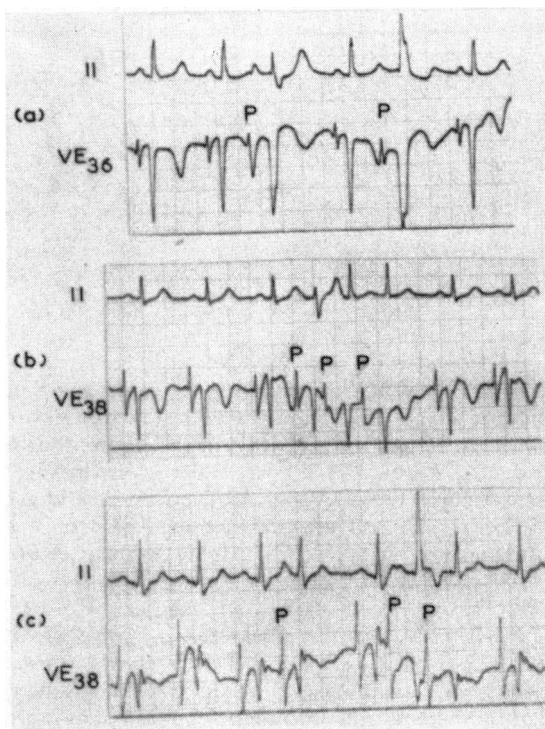


FIG. 1.—Supraventricular premature systoles with aberration of the ventricular complex.

the preceding beat, confusion with ventricular premature systole may occur, and thus the VE lead is particularly helpful.

Lead II (Fig. 2) shows sinus rhythm in the first 3 cycles (rate 100 a minute) followed by sudden halving of the rate for the next 4 cycles. This could be interpreted as a period of 2:1 S-A block; however, inspection of the end of the T waves shows slight terminal negative deflection more apparent in the last two waves, caused by superimposition of premature P waves (nodal). The VE lead clearly demonstrates the presence of 6 premature P waves (nodal), the first 4 being blocked. It is noted that the coupling intervals of the P waves gradually increase until the fifth P wave is conducted with aberrant ventricular complex, while the sixth P wave, with a longer coupling interval, shows a lesser degree of aberration.

*Comment.* Failure of conduction to the ventricle of a premature P wave results in a blocked supraventricular premature systole. Though the degree of prematurity of the P wave is of paramount importance, it is by no means the sole decisive factor. Thus, the observation is not rare that among

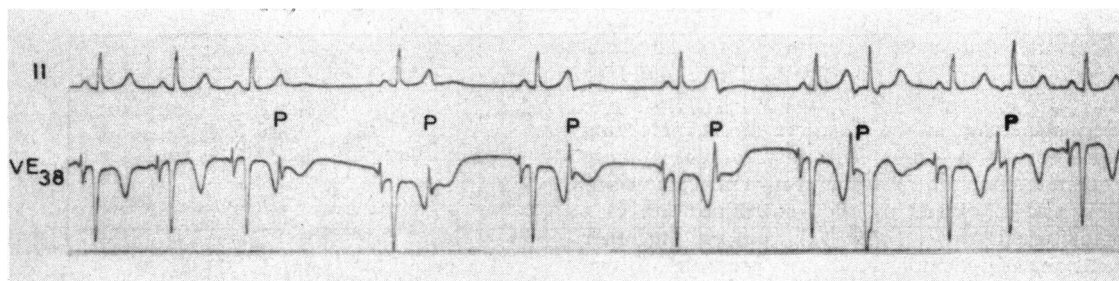


FIG. 2.—Blocked supraventricular premature systoles (nodal) simulating 2:1 sino-atrial block.

premature systoles occurring at the same phase of diastole some are conducted whereas others are blocked (Scherf and Schott, 1953) (see the fourth and fifth P waves in Fig. 2). Variations in vagal tone (Robinson and Draper, 1912), periodic changes of excitability, and the presence or otherwise of a supernormal phase of conduction (Ashman *et al.*, 1925), also play an important part.

*Paroxysmal Atrial Tachycardia with Block.* Lead II (Fig. 3) in the upper strip shows a heart rate of 120 a minute and no discernible P waves. The simultaneously recorded VE lead illustrates the presence of regular P waves at a rate of 225 a minute with identical form and isoelectric intervals between the P waves. The atrial rate is perfectly regular while the ventricular rhythm shows slight irregularity. Though the ventricular response is

essentially explained by the presence of 2:1 atrio-ventricular block, the P waves are seen to march gradually in and out of the QRS complexes. This may represent an occasional Wenckebach response, and explains the slight irregularity of the ventricular rhythm. The ventricular premature systole seen in the upper strip (x) did not disturb the regular atrial mechanism. Though the standard 12-lead electrocardiogram was not helpful in unravelling the nature of arrhythmia present, yet leads to the right of the sternum (V3R in the middle strip) showed the rapid atrial mechanism. The lower strip shows reversion to sinus rhythm which was achieved through continuous potassium infusion for one and a half hours.

*Comment.* Paroxysmal atrial tachycardia with block is an important arrhythmia which is fre-

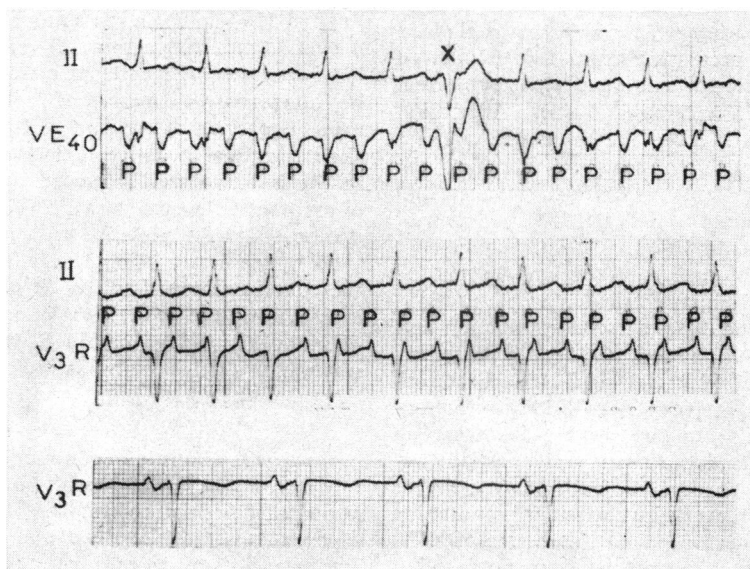


FIG. 3.—Paroxysmal atrial tachycardia with AV heart block (paroxysmal atrial tachycardia with block).

quently the result of digitalis toxicity; prompt diagnosis is important because of its therapeutic implications. In this respect, its differentiation from atrial flutter is important. Digitalis is usually contraindicated in paroxysmal atrial tachycardia with block, but it is the drug of choice in atrial flutter as it slows the ventricular response. The diagnosis is sometimes very difficult to achieve from the standard leads, and here the VE lead is particularly helpful, as it shows rapid P waves with a rate of 150–250 (average 180) a minute, while the rate in atrial flutter generally ranges from 280–310 a minute. The P waves in paroxysmal atrial tachycardia with block usually do not show the sharp intrinsicoid deflection characteristic of the flutter waves, and an isoelectric line is present between the P waves while it is absent in atrial flutter (see Fig. 5).

*Tachycardias of Supraventricular Origin with Aberration of Ventricular Complex.* Leads I and II (Fig. 4) in the upper strips of (A) and (B) show a paroxysmal tachycardia with a rate of 158 a minute. The QRS complexes are widened and notched and no P waves are seen. This could be interpreted as ventricular tachycardia. However, the simultaneously recorded VE lead demonstrates the regular occurrence of P waves with the same rate as the ventricular complexes. The P waves could be interpreted as related to the following ventricular complexes, in which case they represent antegradely conducted P waves; or as related to the preceding ventricular complexes in which case they represent retrogradely conducted P waves. On the basis of the VE lead the tachycardia was presumably interpreted as supraventricular in origin, and intravenous injection of 0.16 mg. deslanoside ("cedilanid")

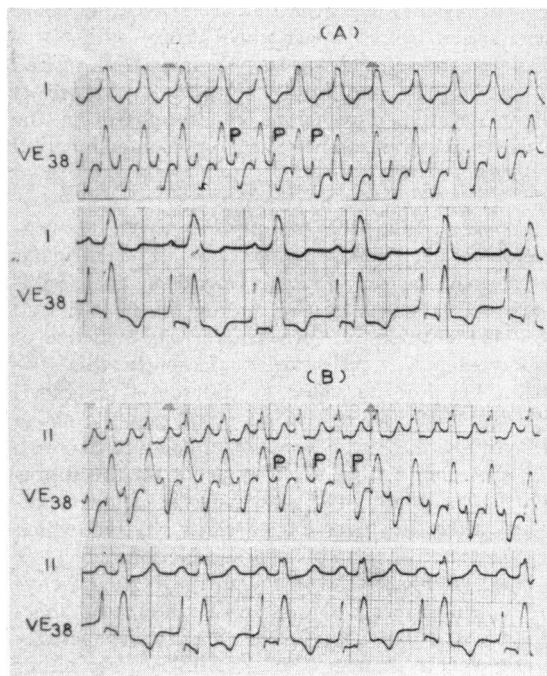


FIG. 4.—Supraventricular tachycardia with widened QRS complexes simulating ventricular tachycardia.

was tried and resulted in interruption of the paroxysm within 3 minutes. Leads I and II in the lower strips of (A) and (B) show regular sinus rhythm with established left bundle-branch block. The VE lead obtained from the same position as during the paroxysm shows the sinus P waves as an upward deflection, in contrast to the downwardly directed ectopic P waves. Therefore

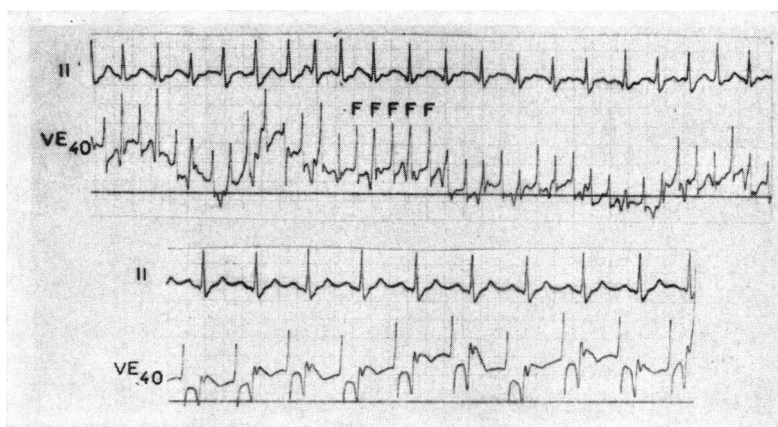


FIG. 5.—Atrial flutter with variable AV block and aberrant ventricular conduction.

the paroxysmal supraventricular tachycardia could have arisen from a lower nodal centre with retrogradely conducted P waves. Re-inspection of lead II during the paroxysm shows the presence of slight negative depressions superimposed on the T waves, which could have well represented inverted P waves.

*Comment.* On the assumption that 1:1 ventriculo-atrial (V-A) conduction in ventricular tachycardia is rare, Rubin, Jagendorf, and Goldberg (1959) said that the finding in the VE lead of a 1:1 relation of QRS and P was evidence of supraventricular tachycardia. Recently this criterion has lost its value in differential diagnosis (Kistin, 1961): there was no proof that the above arrhythmia was not a ventricular tachycardia with 1:1 V-A conduction, the only proof for the supraventricular origin of the tachycardia being established after the reversion to sinus rhythm which demonstrated the established conduction defect.

Lead II (Fig. 5) shows a slightly irregular tachycardia, with average rate of 160 a minute and aberrant ventricular complexes. The atrial activity is not clearly shown. The possibilities are either rapid atrial fibrillation with aberration of the ventricular response, or ventricular tachycardia.

The simultaneously recorded VE lead illustrates the characteristic features of the atrial flutter in the oesophageal electrocardiogram. There are rapid, precisely regular atrial complexes of identical form with sharp intrinsicoid deflection and no iso-electric intervals. The atrial rate is 310 a minute, while the ventricular response averages 160 a minute. There is essentially a 2:1 response in the ventricle; however, the slight irregularity of ventricular rhythm seen in the middle of the tracing is explained by the occasional occurrence of the Wenckebach phenomenon. The lower strip was recorded after reversion to sinus rhythm, and showed an established conduction defect of right bundle-branch block pattern.

V1 in the upper and middle strips (Fig. 6) shows a slightly irregular tachycardia, with an average rate of 110 a minute, and aberrant ventricular complexes. The atrial mechanism is not clearly shown. The case may be interpreted as ventricular tachycardia; but the VE leads taken from different atrial levels did not show independent atrial complexes. On the other hand, they showed the irregular oscillations characteristic of atrial fibrillation. The lower strip shows V1 after slowing of the heart rate; the F waves are readily discernible and the aberrant ventricular complexes have disappeared.

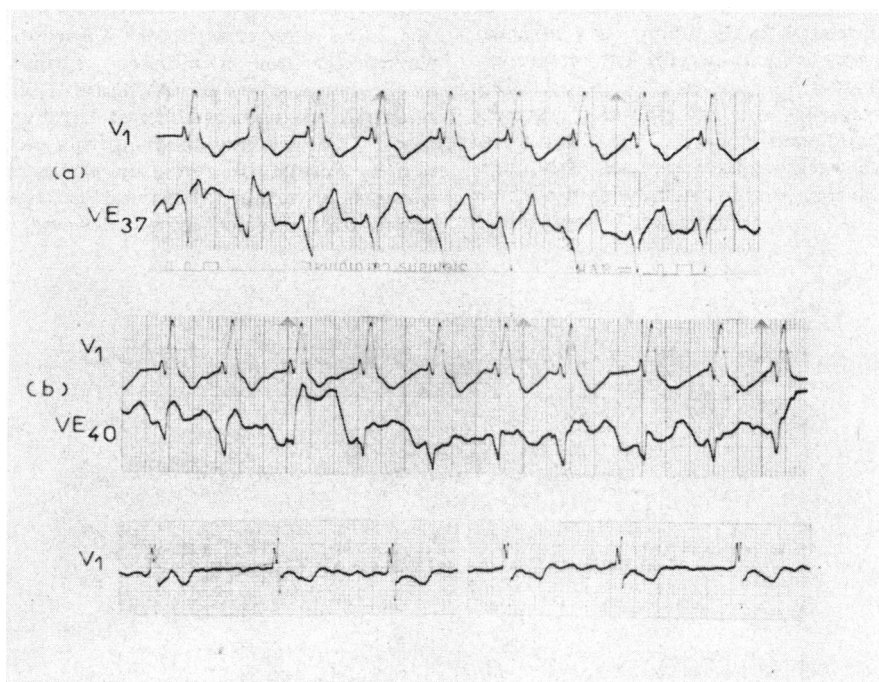


FIG. 6.—Rapid atrial fibrillation with aberration of the ventricular complexes simulating ventricular tachycardia.

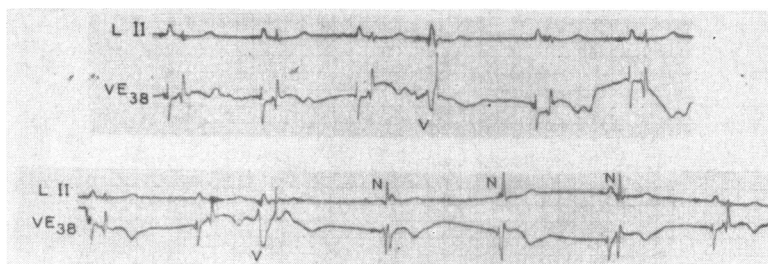


FIG. 7.—Ventricular premature systoles with retrograde conduction resulting in a short run of AV dissociation with aberration of the QRS complex.

*Comment.* The tracings in Fig. 4, 5, and 6 illustrate the importance of the VE lead in diagnosing tachycardias with aberrant ventricular complexes. Many types of paroxysmal tachycardias are considered to be ventricular in origin when they actually arise in the atrium. Tachycardias of supraventricular origin that simulate ventricular tachycardia include any ectopic tachycardia presenting a fairly regular rhythm associated with widened QRS complex. This may occur with atrial or nodal tachycardia, atrial flutter, atrial fibrillation, and occasionally sinus tachycardia with rates of 140 to 180 a minute (Bellet, 1963). In supraventricular tachycardia an abnormal conduction through the ventricle may be due to many factors. Functional fatigue may result in the abrupt or gradual appearance of aberrant ventricular complexes (Vesell and Kraemer, 1951); injury to the conduction system, electrolyte imbalance, digitalis, quinidine, or procainamide hydrochloride therapy may also lead to abnormal QRS complexes (Rubin *et al.*, 1959). In most of these instances the aberration is transitory; however, supraventricular tachycardia may occur on top of an established conduction defect (Bellet, 1963). Certain criteria may help to establish a supraventricular origin; like slowing the heart rate by carotid sinus pressure and/or parasympathomimetic drugs, and the finding of the configuration of the ventricular complex similar to the normal beats of sinus rhythm (Bellet, 1963); the last criterion, however, necessitates the transient reversion to sinus rhythm which is not always available. On the other hand, the VE lead represents a unique tool in unravelling the nature of the atrial mechanism.

*Atrioventricular Dissociation.* In the upper strip (Fig. 7) the fourth ventricular complex is a ventricular premature systole; it is followed by a pause which is not fully compensatory. The VE lead demonstrates ventriculo-atrial (V-A) conduction of the premature systole, with an R-P duration of 0.14 sec. In the lower strip there is a similar

systole, with V-A conduction but with a longer R-P duration of 0.22 sec., so that the negative P wave can be easily seen in lead II. This is followed by three ventricular complexes which differ from the ventricular complexes of the sinus beats in configuration but not in duration (labelled N). In the VE lead sinus P waves are clearly seen immediately after the first aberrant QRS complex, fused with the beginning of the second complex, and immediately preceding the third complex. This is not clearly seen in lead II. This phenomenon represents a short run of AV dissociation with aberrant QRS complex.

The upper and lower strips (Fig. 8) represent a continuous tracing. The nature of the arrhythmia is difficult to elucidate from lead II. On the other hand, 24 P waves are clearly seen in the VE lead and are labelled P1 up to P24. These are of two types: those showing large upward deflection represent sinus P waves, while those with rS deflection are ectopic P waves probably arising from a low atrial or nodal origin (they give rise to negative deflection in lead II). Some of the ectopic P waves are blocked, and some are conducted to the ventricles with varying degree of aberration of the QRS complex. The relatively long pause after the blocked ectopic P9 has initiated a short period of AV dissociation. P10 to P15 are sinus P waves, but they follow the QRS complexes by gradually decreasing R-P intervals. The QRS complexes of the AV dissociation represent nodal escape beats and show slight aberration.

*Comment.* AV dissociation is now recognized to be common (Bellet, 1963). In the usual instances of AV dissociation, interpretation from the standard leads is usually easily made. However, detection of AV dissociation may be difficult in the following instances: (a) when it occurs for a brief interval (for instance after the long pause after S-A heart block, AV heart block, or ventricular premature systole) (Fig. 7); (b) when there is aberration of the ventricular complex of the nodal escape beats



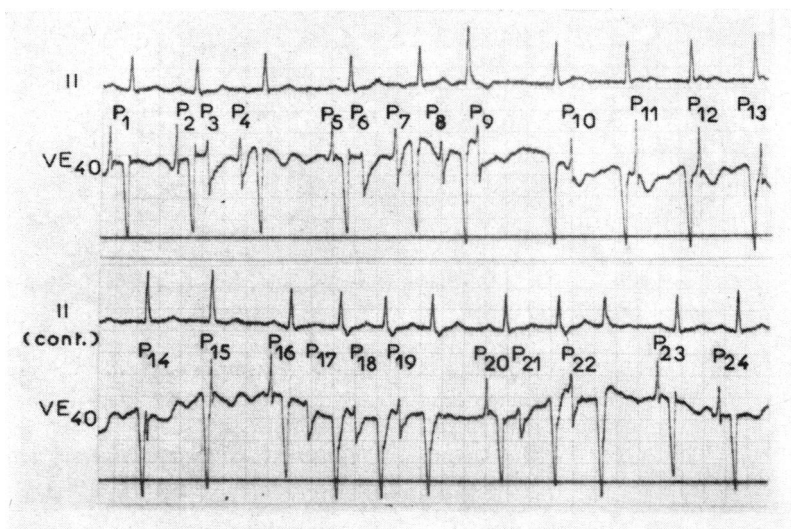


FIG. 8.—Complex arrhythmia showing multiple supraventricular premature systoles (nodal); some are blocked and some are conducted with a period of AV dissociation.

(Fig. 7); and (c) when it occurs in the course of a complex arrhythmia (Fig. 8). In these instances the VE lead is of particular help.

Ventricular aberration of AV nodal escape beats is thought to be rare. It was first reported by Pick (1956), and since then a few more reports have appeared (Walsh, 1962; Kistin, 1966). It has been explained by assuming that impulses arising in peripheral portions of AV junctional tissue, in the vicinity of the point of origin of paraspecific fibres, reach part of the ventricular myocardium over preferential (paraspecific) pathways (Pick, 1956).

*Ventricular Premature Systoles.* Lead II (Fig. 9) shows two systoles: the first is interpolated, and the second is followed by a pause which is not

fully compensatory. The VE lead illustrates the mechanism of the interpolation of the first premature systole, and shows that the ventricular systole which follows this is a response to the regular pacemaking impulse from the S-A node, the normal sequence of the sinus rhythm being undisturbed. The VE lead also explains the non-fully compensatory pause after the second premature systole, and demonstrates a ventriculo-atrial (V-A) conduction of the ventricular premature systole.

The upper strip (Fig. 10) shows three ventricular premature systoles (x, x1, and x2). The VE lead illustrates the atrial mechanism with fully compensatory pause in systole. Note the appearance of sinus P waves as large upward deflections after the QRS complexes of the premature systoles at x and x1. No ventricular complex follows this atrial

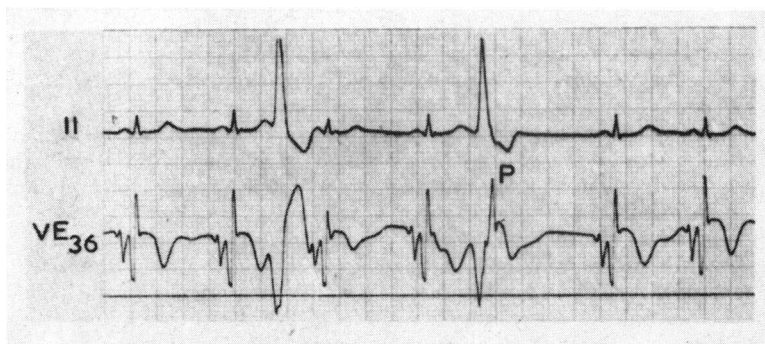


FIG. 9.—Ventricular premature systoles with retrograde conduction and interpolation.

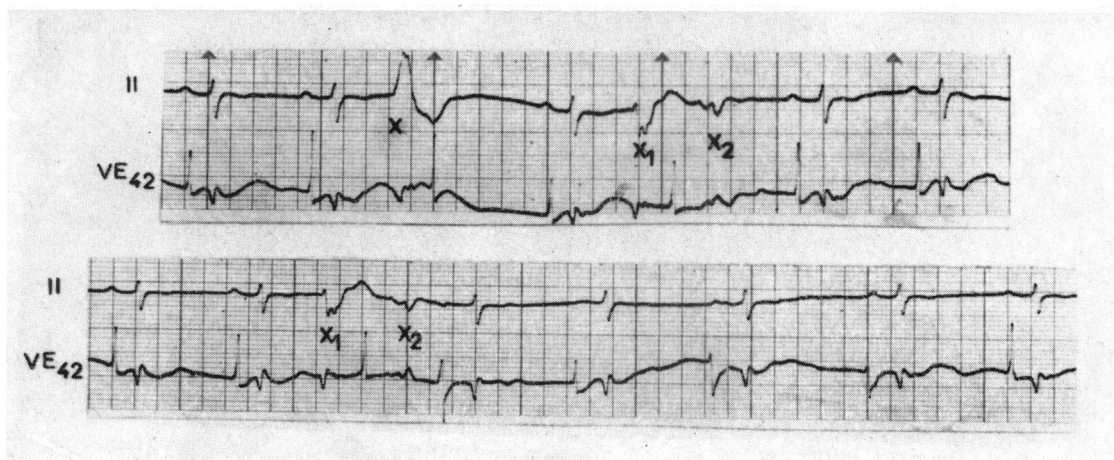


Fig. 10.—Ventricular premature systoles with V-A conduction and reciprocal rhythm.

contraction, because the ventricle is usually in a refractory state at this time. There is no disturbance of the basic atrial rhythm.

In the lower strip a sequence of two premature systoles ( $x_1$  and  $x_2$ ), similar to those in the upper strip, occurred.  $x_1$  is followed by a sinus P wave as in the upper strip; however,  $x_2$  is followed by a retrograde P wave clearly shown in both lead II and the VE lead: this P occurs prematurely to the basic sinus rhythm and is explained by V-A conduction of  $x_2$ ; it is followed by a QRS complex similar in contour to the QRS of sinus rhythm, and is interpreted as a reciprocal beat. This illustrates a second mechanism for the interpolated ventricular premature systole. After the reciprocal beat in response to  $x_2$ , wandering of the pacemaker occurred for the next three beats.

*Comment.* V-A conduction of ventricular premature systoles was in the past considered to be rare; however, in recent years it has been shown to be relatively common (Kistin and Landowne, 1951; Bussan *et al.*, 1955; Kistin, 1959). It is difficult to decide with the standard electrocardiographic leads whether a premature systole is conducted retrogradely or not, for the P waves are usually invisibly hidden in the QRS and T complexes of the premature systole (Bussan *et al.*, 1955). The value of the oesophageal lead in this connexion was particularly emphasized (Kistin and Landowne, 1951; Bussan *et al.*, 1955).

On the other hand, though the interpolated ventricular premature systole was first described in man by Wenckebach as early as 1899, the usual explanation for the ventricular systole which follows the premature systole is that it is a response to the

regular pacemaking impulse from the S-A node. Recently, however, it was shown that some of the systoles that follow the interpolated ventricular premature systoles are reciprocal beats, i.e. they derive their stimulus from the premature systoles (Kistin, 1963). The impulse from the premature systole is conducted to, or towards, the atria, and somewhere above the bifurcation of the bundle of His turns back to activate the ventricle again. This observation supports the hypothesis of multiple pathways of conduction between the atria and ventricles (Moe, Preston, and Burlington, 1956; Rosenblueth, 1958a, b; Kistin, 1959, 1963).

*Paroxysmal Ventricular Tachycardia.* Lead II (Fig. 11) shows a series of 3 or 4 ventricular premature systoles constituting short runs of paroxysmal ventricular tachycardia. In the VE lead the P waves are large downward deflections and can easily be followed through the runs of tachycardia maintaining the sinus rhythm.

Lead II (Fig. 12) shows a series of 20 aberrant ventricular complexes constituting a short paroxysm of ventricular tachycardia. The mechanism of the atrial activation can only be detected from the VE lead.  $x_1$  is shortly preceded by a sinus P wave and represents a fusion beat. Again, a sinus P wave is fused with the descending limb of  $x_2$ . However,  $x_3$  is followed by a retrograde P wave seen as a spiked deflection on the T wave; the V-A conduction time is 0.32 sec.  $x_4$  is again followed by a retrograde P wave, with V-A conduction time of 0.4 sec. In  $x_5$  the retrograde conduction is blocked.  $x_3$ ,  $x_4$ , and  $x_5$  represent the Wenckebach phenomenon. After block of the retrograde conduction in  $x_5$ , the sinus mechanism is again active, and a



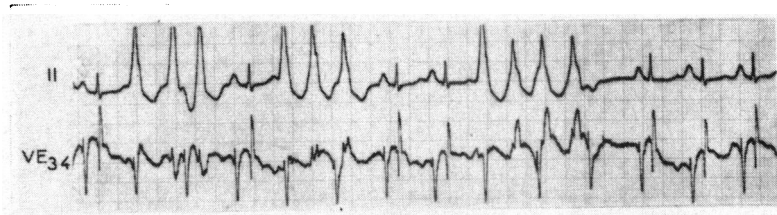


FIG. 11.—Intermittent short runs of paroxysmal ventricular tachycardia with independent atrial rhythm.

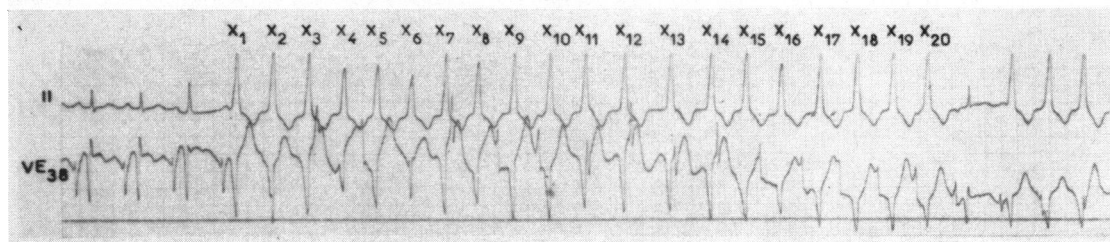


Fig. 12.—Ventricular tachycardia with V-A conduction, Wenckebach phenomenon, and reciprocal rhythm.

sinus P wave is seen fused with the ventricular complex in x6. Retrograde conduction with the Wenckebach phenomenon is repeated once more in the beats x7, x8, x9, and x10 (V-A conduction time 0.32 sec. in x7, 0.42 sec. in x8, 0.5 sec. in x9, and retrograde conduction is blocked in x10). The P wave after x11 is probably a fusion P wave. From x12 to x20 there are retrograde P waves; however, gradual prolongation of the V-A conduction time begins to appear from x15. x20 is followed by a retrograde P wave with the longest V-A conduction time of 0.55 sec., and can be easily seen in lead II; it is followed by a QRS complex similar in contour to the QRS of sinus rhythm and is interpreted as a reciprocal beat.

That the above tachycardia originated in the ventricle and not in the AV node with aberrant conduction is evidenced by the following. (1) The beginning of the paroxysm is recorded and it does not follow a retrograde P wave as would be expected if the origin of the tachycardia is in the A-V node; (2) the tachycardia was induced during catheterization of the right ventricle; and (3) the reciprocal beat has a normal QRS complex. However, the last evidence is not conclusive since the interval from the preceding ectopic beat is longer than the intervals between the ectopic systoles which may allow for recovery from a refractory phase (Kistin, 1961).

*Comment.* Retrograde conduction of paroxysmal ventricular tachycardia was considered rare (Katz

and Pick, 1956). In a review of the clinical literature, only 3 cases of 1:1 V-A conduction and 6 cases of V-A conduction with variable block were found (Foster and Thayer, 1950). Recently, however, Kistin (1961) found V-A conduction in paroxysmal ventricular tachycardia to be a relatively frequent occurrence. He extensively discussed the criteria for the differentiation of the ventricular tachycardia from the AV nodal tachycardia with aberrant conduction. The standard electrocardiographic leads often fail to demonstrate atrial activity accurately. This is apparent from the comparison of lead II with the simultaneously recorded VE lead in Fig. 11 and 12, and probably explains why so few cases of V-A conduction in ventricular tachycardia have been recognized.

*Rare Cardiac Arrhythmias.* In Fig. 13, the 5th ventricular complex is premature and of the same form as the sinus QRS complex. It is closely followed by a rhythmic sinus P wave which is clearly shown in the VE lead and a fully compensatory pause. It is interpreted as a main-stem premature systole. In Fig. 13b the same phenomenon is repeated at x and x1. In Fig. 13c, x is an interpolated main-stem premature systole that resulted in prolongation of the P-R interval of the next sinus beat. x1 is a main-stem premature systole followed by a fully compensatory pause.

*Comment.* Premature systoles arising from the main-stem of the bundle of His are considered to

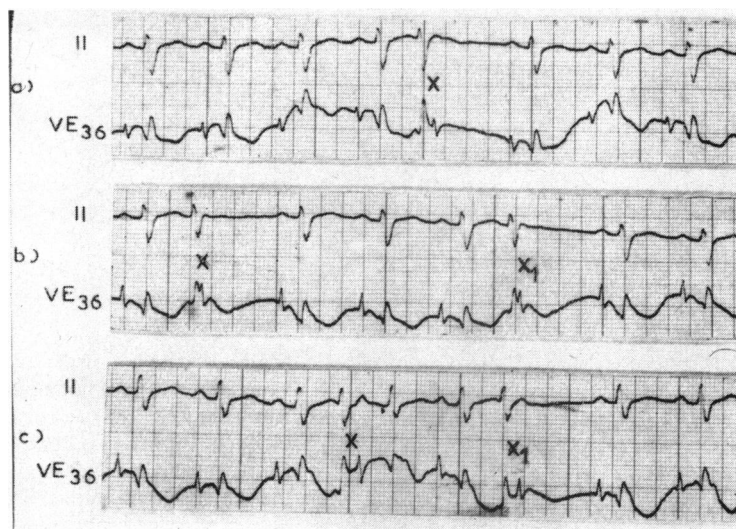


FIG. 13.—Main-stem premature systoles.

be rare (Fletcher, 1955). The criteria considered necessary for the diagnosis of main-stem premature systoles are as follows (Fletcher, 1955): (1) premature beats having the same form as conducted sinus beat, (2) the sequence of P wave undisturbed in time, and (3) a compensatory pause following the premature systole. Marriott and Bradley (1957) reported that main-stem premature systoles can never be diagnosed with certainty, for their diagnosis depends on the assumption that beats originating in the AV node itself would be conducted backwards to the atrium, and that as the beats in question are not so conducted, they are more likely to have arisen at a lower site above the branching of the bundle, i.e. in the main-stem. Recently, however, Marriott and Nizet (1967) have said that the argument on which introduction of the term was based has lost validity, since the frequency of retrograde conduction from even lower (ectopic ventricular) centres has been repeatedly shown, but the term may be conveniently preserved to avoid clumsy circumlocutions.

According to Scherf and Schott (1953), the oesophageal lead will be essential to clarify the atrial mechanism and to establish the diagnosis in main-stem premature systoles; however, oesophageal leads have not yet been recorded in this type of arrhythmia. Fig. 13 represents the only reported case of main-stem premature systoles studied by the VE lead. In addition, it illustrates for the first time the presence of interpolated main-stem premature systoles.

#### DISCUSSION

Though the oesophageal lead was introduced as early as 1906 by Cremer, it was not used much until later (Brown, 1936). However, as late as 1962, a survey conducted by Rodensky and Wasserman disclosed that oesophageal electrocardiography is rarely used in most medical centres. In fact if the electrode is passed with patience and gentleness, the oesophageal lead may be obtained in most patients with little discomfort.

Though the bipolar oesophageal lead (BE) was sometimes found to be superior to the unipolar oesophageal lead (VE) as used in the present study, by cancelling the ventricular deflection while at the same time augmenting the complex of atrial deflection (Copeland *et al.*, 1959), yet we found the VE lead to be satisfactory. Rarely did we encounter the problem of differentiating the superimposed P and QRS complexes, because by obtaining scanning tracings from several atrial levels and the simultaneous drawing of a standard lead recognition of the QRS complex was facilitated. In fact, the use of the BE lead was originally reported by Luisada in 1935, and after this the bipolar oesophageal technique was virtually abandoned until Kistin and Bruce in 1957 drew attention to some of its merits.

However, there are two limitations with the oesophageal lead: firstly, it is sometimes difficult to obtain satisfactory tracings because of wandering of the baseline; and secondly, the oesophageal lead may be difficult to introduce and is poorly tolerated

by severely ill patients. This has led to research in recent years on the use of a simple percutaneous technique for the introduction of an intracardiac electrode, in order to obtain intracavitary right atrial tracings which will have practically the same value as the oesophageal lead (Vogel *et al.*, 1964; Dreifus *et al.*, 1965).

The present study illustrates the value of the oesophageal lead in almost every cardiac arrhythmia. Many of the previously established ideas on cardiac arrhythmias have recently undergone radical changes thanks to the use of oesophageal leads. In this connexion the subject of V-A conduction is particularly germane. Retrograde conduction of ventricular premature systoles or ventricular tachycardia is common in certain animals, but its occurrence in man has, until recently, been considered rare (Scherf and Schott, 1953). The same applies to the problem of reciprocal rhythm. Nowadays, there is both clinical (Wolff, 1959; Kistin, 1959, 1963) and experimental (Moe *et al.*, 1956; Rosenblueth, 1958a, b; Scher *et al.*, 1959) evidence to suggest that the mechanism of V-A conduction and reciprocal rhythm may be in the multiple pathways of AV and V-A conduction.

The relatively common occurrence of 1:1 V-A conduction in ventricular tachycardia makes the problem of differentiation between ventricular tachycardia and supraventricular tachycardia with aberrant conduction more complicated than has been supposed (Kistin, 1961). On the assumption that 1:1 V-A conduction in ventricular tachycardia is rare, the finding in the oesophageal tracing of a 1:1 relation of QRS and P has been used as evidence of supraventricular tachycardia (Rubin *et al.*, 1959). Nowadays, this criterion has lost its value in the differential diagnosis (Kistin, 1961). The interpretation cited for Fig. 4, which was a case of supraventricular tachycardia (nodal) superimposed on established left bundle-branch block, was highly presumptive on the basis of the VE lead alone. There was no proof that the case was not ventricular tachycardia with 1:1 V-A conduction; the only proof in this case and in similar ones could be established only after reversion to sinus rhythm which demonstrated the established conduction defect in the case concerned. We consider this situation to be one of the very few instances where the VE lead cannot offer any help.

The value of the oesophageal lead in unravelling the exact nature of many complex and rare cardiac arrhythmias is exemplified by Fig. 8 and 13. We believe with others (Kistin and Bruce, 1957) that there are many complex arrhythmias that will be recognized more frequently and will be more amenable to analysis by the oesophageal lead. The

routine use of the oesophageal electrocardiographic lead is an indispensable adjunct to elucidate disorders of the heart beat.

#### SUMMARY

A critical assessment of the value of the oesophageal lead in the study of cardiac arrhythmias was carried out on 148 patients with various disturbances of the cardiac rhythm. The VE lead was found helpful in the study of the following cardiac arrhythmias.

- (1) Supraventricular premature systoles in (a) aberration of the ventricular complex simulating ventricular premature systole, and (b) blocked supraventricular premature systoles simulating S-A block.
- (2) Paroxysmal atrial tachycardia with block.
- (3) Tachycardias of supraventricular origin when no P or F waves are discernible in the standard leads, and in the presence of aberration of the ventricular complex simulating ventricular tachycardia.
- (4) A-V dissociation in the following instances; (a) when it occurs for a brief interval, (b) when there is aberration of the ventricular complex of the nodal escape beats, and (c) when it occurs in the course of a complex arrhythmia.
- (5) In the study of ventricular premature systoles the VE lead has elucidated many of the mechanisms concerned with V-A conduction and the reciprocal rhythm. It gives evidence on the existence of multiple pathways of AV and V-A conduction.
- (6) In ventricular tachycardia the VE lead was indispensable in unravelling the nature of atrial activation. It demonstrated the frequent occurrence of V-A conduction in ventricular tachycardia.
- (7) The VE lead is of great value in unravelling the nature of rare and complex cardiac arrhythmias.

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